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NEWS
                 "Ask CAS" for self-help around the clock
      2
NEWS
                 The Derwent World Patents Index suite of databases on STN
         OCT 23
                 has been enhanced and reloaded
NEWS
         OCT 30
                 CHEMLIST enhanced with new search and display field
NEWS
         NOV 03
                 JAPIO enhanced with IPC 8 features and functionality
         NOV 10
NEWS
                 CA/CAplus F-Term thesaurus enhanced
     6
         NOV 10
                 STN Express with Discover! free maintenance release Version
NEWS
                 8.01c now available
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NEWS
                 CA/CAplus to MARPAT accession number crossover limit increased
                 to 50,000
         DEC 01
                 CAS REGISTRY updated with new ambiguity codes
NEWS
     9
NEWS 10
         DEC 11
                 CAS REGISTRY chemical nomenclature enhanced
         DEC 14
NEWS 11
                 WPIDS/WPINDEX/WPIX manual codes updated
NEWS 12
         DEC 14
                 GBFULL and FRFULL enhanced with IPC 8 features and
                 functionality
                 CA/CAplus pre-1967 chemical substance index entries enhanced
NEWS 13
         DEC 18
                 with preparation role
         DEC 18
NEWS 14
                 CA/CAplus patent kind codes updated
         DEC 18
NEWS 15
                 MARPAT to CA/CAplus accession number crossover limit increased
                 to 50,000
         DEC 18
NEWS 16
                 MEDLINE updated in preparation for 2007 reload
         DEC 27
NEWS 17
                 CA/CAplus enhanced with more pre-1907 records
NEWS 18
         JAN 08
                 CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS 19
         JAN 16
                 CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS 20
         JAN 16
                 IPC version 2007.01 thesaurus available on STN
         JAN 16
NEWS 21
                 WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS 22
         JAN 22
                 CA/CAplus updated with revised CAS roles
NEWS 23
         JAN 22
                 CA/CAplus enhanced with patent applications from India
NEWS 24
         JAN 29
                 PHAR reloaded with new search and display fields
NEWS 25
         JAN 29
                 CAS Registry Number crossover limit increased to 300,000 in
                 multiple databases
NEWS 26
         FEB 13
                 CASREACT coverage to be extended
NEWS 27
         Feb 15
                 PATDPASPC enhanced with Drug Approval numbers
NEWS 28
         Feb 15
                 RUSSIAPAT enhanced with pre-1994 records
NEWS 29
         Feb 23
                 KOREAPAT enhanced with IPC 8 features and functionality
NEWS 30
         Feb 26
                 MEDLINE reloaded with enhancements
NEWS 31
         Feb 26
                 EMBASE enhanced with Clinical Trial Number field
NEWS 32
         Feb 26
                 TOXCENTER enhanced with reloaded MEDLINE
NEWS 33
         Feb 26
                 IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS 34
         Feb 26
                 CAS Registry Number crossover limit increased from 10,000
                 to 300,000 in multiple databases
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NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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=> (autism or autistic) and (dipeptideyl peptidase or CD26 or CD 13 or CD69 or CD 69 or CD13 or CD 26)

L1 0 FILE AGRICOLA

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ANSWER 1 OF 3 LIFESCI COPYRIGHT 2007 CSA on STN

ACCESSION NUMBER: 2005:1197 LIFESCI

TITLE: Heat Shock Protein and Gliadin Peptide Promote Development

of Peptidase Antibodies in Children with Autism

and Patients with Autoimmune/Disease

Vojdani, A.*; Bazargan, M.; Vojdani, E.; Samadi, J.; Nourian, A.A.; Eghbalieh, N.; Cooper, E.L. AUTHOR:

CORPORATE SOURCE: Section of Neuroimmuno ogy, Immunosciences Lab., Inc., 8693

Wilshire Blvd., Suite 200, Beverly Hills, CA 90211; E-mail:

4/4/12/12

immunsci@ix.netcom.com

Clinical and Diagrostic Laboratory Immunology [Clin. Diagn. SOURCE:

Lab. Immunol.], (20040500) vol. 11, no. 3, pp. 515-524.

ISSN: 1071-412X

DOCUMENT TYPE: Journal

FILE SEGMENT:

LANGUAGE: English SUMMARY LANGUAGE: English

Searching for a mechanism underlying autoimmunity in autism, we postulated that gliadin peptides, heat shock protein 60 (HSP-60), and streptokinase (SK) bind to different peptidases resulting in autoantibody production against these components. We assessed this hypothesis in patients with autism and in those with mixed connective tissue diseases. Associated with antigliadin and anti-HSP antibodies, children with autism and patients with autoimmune disease developed anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV (or CD26)) and anti-aminopeptidase N (CD13) autoantibodies. A significant percentage of autoimmune and autistic sera were associated with elevated immunoglobulin G (IgG), IgM, or IgA antibodies against three peptidases, gliadin, and HSP-60. These antibodies are specific, since immune absorption demonstrated that only specific antigens (e.g., DPP IV absorption of anti-DPP IV), significantly reduced IgG, IgM, and IgA antibody levels. For direct demonstration of SK, HSP- 60, and gliadin peptide binding to DPP IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and antigliadin antibodies. Adding SK, HSP-60, and gliadin peptides to DPP IV resulted in 27 to 43% inhibition of the DPP IV-anti- DPP IV reaction, but DPP IV-positive peptides caused 18 to 20% enhancement of antigen-antibody reactions. We propose that (i) superantigens (e.g., SK and HSP- 60) and dietary proteins (e.g., gliadin peptides) in individuals with predisposing HLA molecules bind to aminopeptidases and (ii) they induce autoantibodies to peptides and tissue antigens. Dysfunctional membrane peptidases and autoantibody production may result in neuroimmune dysregulation and autoimmunity.

ACCESSION NUMBER: 2004:108019 LIFESCI

TITLE: Infections, toxic chemicals and dietary peptides binding to

lymphocyte receptors and tissue enzymes are major

instigators of autoimmunity in autism

AUTHOR:

Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L. CORPORATE SOURCE: 8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211,

USA; E-mail: DrAri@msn.com

SOURCE: International Journal of Immunopathology and Pharmacology

[Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no.

3, pp. 189-199. ISSN: 0394-6320.

DOCUMENT TYPE: Journal

FILE SEGMENT:

LANGUAGE: English SUMMARY LANGUAGE: English

Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-ethyl mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and ethyl mercury binding to CD26 and CD69, indicating that they are specific.

Immune absorption demonstrated that only specific antigens, like

CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin,

casein and ethyl mercury to CD26 or CD69, microtiter

wells were coated with CD26 or CD69 alone or in

combination with SK, gliadin, casein or ethyl mercury and then reacted

with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-

CD26 or anti-CD69 antibodies. The highest % binding of

these antigens or peptides to CD26 or CD69 was

attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl mercury) in individuals with pre-disposing HLA

molecules, bind to CD26 or CD69 and induce antibodies against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in

autoimmune reaction in children with autism.

ANSWER 3 OF 3 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN L9 DUPLICATE

ACCESSION NUMBER: 2003:36798209 BIOTECHNO

TITLE: Opioid peptides and dipeptidyl peptidase in

autism

AUTHOR: Hunter L.C.; O'Hare A.; Herron W.J.; Fisher L.A.;

Jones G.E.

CORPORATE SOURCE: A. O'Hare, Royal Hospital for Sick Children, Community

Child Health Services, 10 Chalmers Crescent, Edinburgh

EH9 1TS, United Kingdom. E-mail: AO'Hare@ed.ac.uk

SOURCE: Developmental Medicine and Child Neurology, (01 FEB

2003), 45/2 (121-128), 40 reference(s)

CODEN: DMCNAW ISSN: 0012-1622

DOCUMENT TYPE:

Journal; Article

COUNTRY: LANGUAGE: United Kingdom

English | English

SUMMARY LANGUAGE:

BIOTECHNO

AB

2003:36798209 It has been hypothesized that autism results from an 'opioid peptide excess'. The aims of this study were to (1) confirm the presence of opioid peptides in the urine of children with autism and (2) determine whether dipeptidyl peptidase IV (DPPIV/CD26) is defective in children with autism. Opioid peptides were not detected in either the urine of children with autism (10 children; nine males, one female; age range 2 years 6 months to 10 years 1 month) or their siblings (10 children; seven males, three females; age range 2 years 3 months to 12 years 7 months) using liquid chromatography-ultraviolet-mass spectrometric analysis (LC-UV-MS). Plasma from 11 normally developing adults (25 years 5 months to 55 years 5 months) was also tested. The amount and activity of DPPIV in the plasma were quantified by an ELISA and DPPIV enzyme assay respectively; DPPIV was not found to be defective. The percentage of mononuclear cells expressing DPPIV (as CD26) was determined by flow cytometry. Children with autism had a significantly lower percentage of cells expressing CDS and CD26, suggesting that they had lower, T-cell numbers than their siblings. In conclusion, this study failed to replicate the findings of others and questions the validity of the opioid peptide excess theory for the cause of autism.